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Symbol	Name	Synonyms	Organism
ERBB3	v-erb-b2 erythroblastic leukemia viral oncogene homolog 3 (avian)	c-erbB3, c-erbB-3, ErbB-3, erbB3-S, HER3, MDA-BF-1, MGC88033, p180-ErbB3, p45-sErbB3, p85-sErbB3, Receptor tyrosine-protein kinase erbB-3 precursor, Tyrosine kinase-type cell surface receptor HER3	Homo sapiens

UniProt	P21860, Q9BUD7, Q9NNX2
IntAct	P21860
PDB Structure	1M6B
OMIM	190151
NCBI Gene	2065
NCBI RefSeq	NP_001973, NP_001005915
NCBI RefSeq	NM_001982, NM_001005915
NCBI UniGene	2065
NCBI Accession	S61953, CR621450

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Human epidermal growth factor receptor-3 (**HER3**) is a member of the type I receptor tyrosine kinase family. [2003]

Heregulin (HRG) belongs to a family of polypeptide growth factors that bind to receptor tyrosine kinases **ErbB3** and ErbB4. [2001]

Concept & Implementation by Robert Hoffmann

The four receptor tyrosine kinase I receptors, ErbB-1, ErbB-2, **ErbB-3**, and ErbB-4, which have been implicated in the development of a variety of normal and malignant tissues, are activated through ligand mediated homo- and heterodimerization. [1998]

The **ErbB3** protein is a member of the ErbB subfamily of receptor protein tyrosine kinases. [1994]

The processes by which **ErbB-3**, an inactive tyrosine kinase, exerts its biological effects are poorly understood. [2000]

ErbB-3 and ErbB-4 are the most recently discovered and least characterized of the class I tyrosine kinase receptors. [1997]

ErbB-3 is noteworthy for its low tyrosine kinase activity, suggesting that it may function more as an adaptor in signaling than as a kinase. [1997]

ErbB3 lacks tyrosine kinase activity. [2002]

Several mutations in the tyrosine kinase domain of **ErbB-3** have been postulated to render this enzyme catalytically inactive. [1998]

c-erbB3 gene encodes secreted as well as transmembrane receptor tyrosine kinase. [1993]

Escape from HER-family tyrosine kinase inhibitor therapy by the kinase-inactive **HER3**. [2007]

The protein product of the member of the epidermal growth factor receptor family **ERBB3** could be shown to be highly present in all of the CCSST cell lines investigated, as well as in 18 of 20 primary tumor biopsies. [2004]

We have examined the expression of the two **ERBB3** transcripts by Northern blotting in cancer cell lines and normal human fetal and adult tissues. [2001]

Recombinant neuregulin-2beta induces the tyrosine-phosphorylation of ErbB2, **ErbB3** and ErbB4 in cell lines expressing all of these ErbB-family receptors. [1997]

Isolation and characterization of four alternate **c-erbB3** transcripts expressed in ovarian carcinoma-derived cell lines and normal human tissues. [1998]

Transcriptional regulation of the **c-erbB-3** gene in human breast carcinoma cell lines. [1993]

In this paper we describe the cloning of the **c-erbB-3** promoter and its functional analysis within mammary-derived cell lines. [1993]

Our studies found that ErbB2 and **ErbB3** were expressed by each of these cell lines. [2004]

ErbB-3 expression and activation did not follow a consistent pattern between cell lines. [2006]

Overexpression of **c-erbB-3 [?]** and c-erbB-4 was more frequently observed in newly derived HNSCC lines than in long-established cell lines. [2001]

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ErbB2 overexpression caused tyrosine phosphorylation of ErbB2, whereas **ErbB3** and ErbB4 were only slightly tyrosine phosphorylated. [2005]

The transfected cells showed inactivation of ErbB2 tyrosine phosphorylation and reduced heterodimerization of ErbB2 and **ErbB3**. [2005]

The fatty acid enhanced the tyrosine phosphorylation of ErbB4 but not of ErbB2 or **ErbB3**. [2001]

NRG1beta preferentially acts through **ErbB3** in these cells by stimulating the tyrosine phosphorylation and recruitment of phosphatidylinositol 3-kinase and Shc to that receptor. [1998]

In MDA-MB-453 cells, both NRG1beta and NRG2beta stimulate the tyrosine phosphorylation of the ErbB2 and **ErbB3** receptors to similar extents, but only NRG1beta potently stimulates morphological changes consistent with their differentiation. [1998]

ErbB2 and **ErbB3** do not affect the amount of ligand-induced ErbB4 tyrosine phosphorylation. [2002]

ErbB2 and **ErbB3** do not quantitatively modulate ligand-induced ErbB4 tyrosine phosphorylation. [2002]

Although both NTAKs induce the highest level of tyrosine phosphorylation of ErbB2, NTAKalpha2a and NTAKbeta preferentially induce **ErbB3** and ErbB4 phosphorylation, respectively. [2000]

To identify novel molecular changes associated with breast cancer progression, we conducted phosphoproteomics of the MCF10AT model comprising isogenic, ErbB2- and **ErbB3**-positive, xenograft-derived cell lines that mimic different stages of breast cancer. [2007]

Overexpression of ErbB1, ErbB2 and ErbB4 receptors was enough to activate them as judged by their phosphorylation, whereas co-expression of other ErbB receptors was necessary for the phosphorylation of the **ErbB3**. [2000]

Heregulin (HRG) is a soluble secreted growth factor, which, upon binding and activation of **ErbB3** and ErbB4 transmembrane receptor tyrosine kinases, is involved in cell proliferation, invasion, survival and differentiation of normal and malignant tissues. [2007]

Expression of the **ERBB3** gene product in breast cancer. [1992]

Binding of heregulin (HRG) to its receptor, **ErbB3**, results in a dimerization with ErbB2/neu and activation of their intrinsic tyrosine kinases, initiating a cascade of events resulting in the stimulation of acetylcholine receptor (AChR) genes in muscle. [1997]

As in the transfected cells, purified Hst inhibited **HER-3** levels and suppressed HRG-induced proliferation of MCF7 and BT474 breast cancer cells. [2003]

These observations suggest that overexpression of **c-erbB3** protein could play an important role in tumour progression from non-invasive to invasive and, also, that it may have the potential to be used as a marker for poor prognosis of breast cancer. [1998]

The secreted/shed NRG-1alpha was capable of activating the ErbB2/**ErbB3** receptor complex expressed on the breast adenocarcinoma cell line MCF-7. [2004]

A flow cytometric study of **c-erbB-3** expression in breast cancer. [1995]

Essential function for **ErbB3** in breast cancer proliferation. [2004]

Now, in a recent report, it has been shown that **ErbB3** is a critical partner for the transforming activity of ErbB2 in breast cancer cells. [2004]

In fact, abundant **ErbB-3** expression is detected only in gefitinib-sensitive NSCLC cell lines. [2005]

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Two malignant melanoma cell lines expressed ErbB2 and **ErbB3**, but not the full-length ErbB4 receptor. [2005]

METHODS AND FINDINGS: Using human breast cancer cell lines displaying different levels of alpha6beta4 and **ErbB-3** receptors and a series of 232 breast cancer biopsies from patients submitted to adjuvant Tamoxifen monotherapy for five years, we evaluated the functional interaction between both receptors in relationship to Tamoxifen responsiveness. [2008]

Overexpression of **ERBB3** appears to result from increased levels of gene transcription since in none of the cell lines or primary cancers analysed did we find evidence of gene amplification. [1992]

In this study, we found that elevated levels of **ErbB-3** expression did not occur in the absence of AP-2gamma in a panel of human mammary epithelial and fibroblasts cell lines. [2002]

Neuregulins (NRGs), which are highly expressed in the nervous system, bind and activate two receptor tyrosine kinases, **ErbB-3** and ErbB-4. [2005]

A panel of human glioma cell lines, which had previously been analyzed for ErbB-2 expression, was examined for **ErbB-3** and ErbB-4 expression. [1997]

In this study, we purified the 200 kDa protein from an extract of NUGC-4 cells, a cell line of signet-ring cell carcinoma, and identified it as **ErbB3**. [2003]

HER-3 was expressed in all rhabdomyosarcoma cell lines. [2000]

ERBB3 kinase domain mutations are rare in lung, breast and colon carcinomas. [2006]

Taken together, activation of **ErbB3** pathway may contribute to the development of dedifferentiated carcinomas. [2003]

Expression of **c-erbB3** protein in primary breast carcinomas. [1998]

Immunocoprecipitation and Western blot studies indicated that beta-HCH mainly promotes dimerization of ErbB2 and **ErbB3** at early time points, whereas HRG causes their dimerization with a rapid and significant rise in their tyrosine phosphorylation levels. [2002]

Thus, the ErbB2/**ErbB3** dimer functions as an oncogenic unit to drive breast tumor cell proliferation. [2003]

Heregulins are a family of ligands for the **ErbB3**/ErbB4 receptors that play important roles in breast cancer cell proliferation and tumorigenesis. [2006]

Inactivation of **ErbB3** by siRNA promotes apoptosis and attenuates growth and invasiveness of human lung adenocarcinoma cell line A549. [2005]

Conjugated linoleic acid inhibits cell proliferation and **ErbB3** signaling in HT-29 human colon cell line. [2003]

ErbB3 was found to be phosphorylated selectively in dedifferentiated adenocarcinoma cell lines among various gastric cancer cell lines. [2003]

In this report we demonstrate by nucleic acid analysis and immunohistochemistry that the recently recognised third member of this gene family, **ERBB3**, shows a wide range of expression in breast cancer, and shows stronger immunoreactivity than that observed in normal tissue in 43 out of 195 cases (22%) of primary breast cancer. [1992]

Despite its high affinity for ErbB1 and **ErbB3**, this mutant was unable to activate ErbB1.ErbB3 heterodimers, as shown by the cell survival and receptor phosphorylation analysis. [2006]

In mammary carcinoma cells, we evidenced that the alpha6beta4 integrin strongly influence Akt phosphorylation through **ErbB-3** protein regulation. [2008]

top

Inactivation of Akt by the epidermal growth factor receptor inhibitor erlotinib is mediated by **HER-3** in pancreatic and colorectal tumor cell lines and contributes to erlotinib sensitivity. [2006]

Expression profiling of t(12;22) positive clear cell sarcoma of soft tissue cell lines reveals characteristic up-regulation of potential new marker genes including **ERBB3**. [2004]

We also found that the association between **ErbB-3** and P-Akt positivity mainly occurs in ERbeta1-negative breast cancer derived from patients with lower DFS indicating that both receptors are clinically relevant in predicting the response to Tamoxifen. [2008]

c-erbB3 and c-erbB4 expression is a feature of the endocrine responsive phenotype in clinical breast cancer. [1998]

Moreover, the **ErbB-3** inactivation inhibits Akt phosphorylation, induces apoptosis and inhibits in vitro invasion favouring Tamoxifen responsiveness. [2008]

Knockdown of **HER-3** in BxPC3, an erlotinib-sensitive pancreatic tumor cell line, results in inhibition of the phosphorylation for both Akt and S6 and is associated with a decrease in cell proliferation and reduced sensitivity to erlotinib. [2006]

By infection of ErbB2-overexpressing breast cancer cells with a retrovirus expressing E3, we show that **ErbB3** is an essential partner in the transformation process. [2003]

Cell proliferation was increased when ErbB2 and **ErbB3** were both overexpressed. [2005]

It was found that epithelial cells in primary PCa did not express **MDA-BF-1**. [2004]

We have previously shown that HRG-beta is mitogenic for various human mammary epithelial cell lines that coexpress c-erbB-2 and **c-erbB-3**. [2000]

The **c-erbB-3** protein is expressed in normal breast epithelial cells and has been reported to be present at high levels in some cancers but at normal levels or at lower than normal levels in some others. [1998]

The type 1 family of growth factor receptors, which consist of the epidermal growth factor receptor, c-erbB-2, **c-erbB-3**, and c-erbB-4, are expressed in normal breast ductal epithelial cells and in some breast cancers. [1998]

Moreover, the addition of recombinant heregulin or antibodies capable of disrupting ErbB-2/**ErbB-3** complexes had no effect on cell proliferation. [1997]

Clinical and prognostic significance of the expression of the c-erbB-2 and **c-erbB-3** oncoproteins in primary and metastatic malignant melanomas and breast carcinomas. [1997]

Heregulins (HRG) are known as soluble secreted growth factors that, on binding and activating **ErbB3** and ErbB4 cell surface receptors, are involved in cell proliferation, metastasis, survival, and differentiation in normal and malignant tissues. [2006]

Suppression of **ErbB3** expression reduced both intravasation and metastasis. [2006]

A series of 549 cases of primary lung carcinomas were immunostained with a monoclonal anti-human **c-erbB-3** antibody (Clone RTJ.1) using formalin-fixed, paraffin-embedded archival tissue. [1997]

In this systematic immunocytochemical study we observed the expression (overexpression) of the c-erbB-2 and **c-erbB-3** oncoproteins in 30 primary cutaneous malignant melanomas (CMMs), 10 already metastasized malignant melanomas (MMMs) and 15 lymph-node negative breast carcinomas (BCs). [1997]

In biologically relevant systems, interaction of HRG with **ErbB3** or ErbB4 results in the transactivation of ErbB2. [1996]

The **ErbB3** receptor and the downstream signaling kinase Akt are implicated in proliferation of lung adenocarcinoma cells. [2005]

top

It was found that the overexpression of **c-erbB3** protein was observed in 67% (6/9) of comedo DCIS, 52% (44/84) of invasive ductal carcinomas, 71% (5/7) of carcinomas containing both the in situ and invasive lesions and 25% (1/4) of invasive lobular carcinomas. [1998]

Expression of **c-erbB3** protein was investigated in 104 primary breast carcinomas comprising nine comedo ductal carcinoma in situ (DCIS), 91 invasive ductal carcinomas and four invasive lobular carcinomas using two monoclonal antibodies, RTJ1 and RTJ2. [1998]

These same transcripts were also detected in **ErbB-3** overexpressing human tumor cell lines derived from breast and lung carcinomas, and a sarcoma. [1999]

Induction of **ErbB-3** Expression by alpha6beta4 Integrin Contributes to Tamoxifen Resistance in ERbeta1-Negative Breast Carcinomas. [2008]

High expression of **ERBB3** is positively associated with the presence of lymph node metastases, but there was no demonstrable relationship with patient survival in this series. [1992]

Prognostic significance of **ERBB3** overexpression in oral squamous cell carcinoma. [1995]

These results demonstrated that **ERBB3** expression may be helpful in identifying those oral squamous cell carcinomas with higher malignant potential. [1995]

Through their interaction with the ErbB family of receptors (ErbB2, **ErbB3** and ErbB4), neuregulins help to regulate cell growth and differentiation in many tissues. [1997]

Loss of functional ErbB2 or **ErbB3** has similar effects on cell proliferation and cell cycle regulators. [2003]

Instead, the disruption of **HER3** oligomers by hrg results in an approximately 10-fold increase in total binding sites, but the newly created binding sites are of lower affinity. [2003]

With ATFs, the effects of changes in ErbB2 and **ErbB3** receptor levels were evaluated by using cell proliferation, cell migration, and cell signaling assays. [2005]

Cell growth assays show that exogenous addition of a 100-fold molar excess of **p85-sErbB3** inhibits HRG-stimulated cell growth by as much as 90%. [2001]

Together these results suggest that **p85-sErbB3** is a naturally occurring negative regulator of HRG-stimulated signal transduction that may have important therapeutic applications in human malignancies associated with HRG-mediated cell growth such as breast and prostate cancer. [2001]

Identification of a heregulin binding site in **HER3** extracellular domain. [2001]

Next, we identified a putative hrg binding site by limited proteolysis of the recombinant extracellular domains of **HER3** (HER3-ECD(I-IV)) in both the presence and absence of hrg. [2001]

ErbB2 and **ErbB3** receptors mediate inhibition of calcium-dependent chloride secretion in colonic epithelial cells. [1999]

In A549 cells, **ErbB3** is indicated as having major effects on cell division, survival, motility, migration and invasiveness. [2005]

Immunoprecipitation analysis of primary cultures of human ovarian carcinomas also demonstrate the expression of a 90 kDa **ErbB-3** related protein that is secreted. [1998]

Dominant negative interference of transcription factor AP-2 causes inhibition of **ErbB-3** expression and suppresses malignant cell growth. [2002]

ErbB-3 predicts survival in ovarian cancer. [2006]

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CONCLUSION: **HER3** may represent a new prognostic factor in primary epithelial ovarian cancer. [2006]

Here, we studied a possible association between **HER3** expression and prognosis in patients with ovarian cancer. [2006]

METHODS: Tumor tissue of 116 consecutive patients diagnosed with primary epithelial ovarian cancer between 1986 and 1995 was analyzed immunohistochemically for **HER3** expression. [2006]

In contrast, ligand binding facilitates heterodimerization with ErbB2 and is expected to stabilize an extended conformation of the **ErbB3** extracellular domain (ECD) in which the dimerization interface is exposed. [2005]

In contrast, epithelial cells in 41 of 45 PCa metastases (18 of 19 lymph node metastases and 23 of 26 bone metastases), and activated osteoblasts in bone metastases, expressed **MDA-BF-1**. [2004]

Neither **c-erbB-3** nor c-erbB-4 had any association with survival. c-erbB-2 had an independent prognostic effect on overall and disease-free survival (DFS) in all cases, as well as in the subset of breast carcinoma patients with nodal metastases. [2004]

Twenty-eight invasive ductal carcinomas and 1 DCIS were stained for **c-erbB-3** expression: 2 were grade I (Bloom and Richardson), 15 grade II, and 11 grade III tumours, 1 being unclassified; 16 were axillary node positive and 10 node negative; in 2 cases no nodes were sampled. [1995]

Since ErbB receptor is important for growth, metastasis and drug resistance, inhibition of ErbB-2 and **ErbB-3** by pharmacological doses of quercetin may provide a new approach for treatment of prostate cancers. [2003]

Morphogenetic and proliferative responses to heregulin of mammary epithelial cells in vitro are dependent on HER2 and **HER3** and differ from the responses to HER2 homodimerisation or hepatocyte growth factor. [2000]

Ionizing radiation stimulates existing signal transduction pathways involving the activation of epidermal growth factor receptor and **ERBB-3**, and changes of intracellular calcium in A431 human squamous carcinoma cells. [1999]

The strongest staining for ErbB2 and **ErbB3** was observed in the superficial ocular surface epithelium. [2001]

These studies suggest that **HER-3** could be used as a biomarker to select patients who are most likely to respond to erlotinib therapy. [2006]

Markedly elevated **ERBB3** mRNA levels were demonstrated in certain human mammary tumor cell lines. [1989]

We found the avian erythroblastic leukemia viral oncogene homologue 3 (**ERBB3**) to be one of the most dramatically up-regulated genes in CCSST. [2004]

High expression of erbB3 protein (**ERBB3**) was significantly associated with lymph node metastasis ($P < 0.05$), survival rate ($P < 0.05$) and mode of invasion ($P < 0.01$) in this series. [1995]

HRG binding induces **ErbB3** and ErbB4 heterodimerization with ErbB2, activating downstream signal transduction. [2001]

To investigate the selectivity in dimer formation by ligands, we have applied the phage display approach to obtain ligands with modified C-terminal residues that discriminate between ErbB-2 and **ErbB-3** as dimerization partners. [2003]

Phosphatidylinositol (PI) 3-kinase, an enzyme believed to be important in cellular growth regulation by growth factors and oncogenes, is predicted to couple to tyrosine-phosphorylated **ErbB3**. [1995]

RESULTS: Expression levels of pErbB2 ($P = 0.02$) and total **ErbB3** protein ($P = 0.02$) associated with resistance to gefitinib. [2006]

ErbB2 and **ErbB3** may have potential as predictive markers and as therapeutic targets for combination therapy in treatment of HNSCC with gefitinib. [2006]

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Expression of the **c-erbB3** protein was determined in transitional cell carcinoma of the bladder by immunohistochemistry. [1996]

ErbB2 and **ErbB3** have been implicated in the development of colon cancer, and both proteins are expressed at high levels in the HT-29 cell line. [2003]

To examine CLA regulation of HT-29 cell proliferation and apoptosis and the influence of CLA on the **ErbB3** signaling pathway, HT-29 cells were cultured in the presence of CLA and/or heregulin. [2003]

In all stages, squamous cell carcinoma showed the greatest rate of high **c-erbB-3** positivity (score, 3) (34/119; 28.6%), followed by adenocarcinoma (41/256; 15.9%) and large cell carcinoma (7/66; 10.6%). [1997]

Expression of the oncogenes c-erbB-2 and **cerbB-3** was very low in adenosis, and the staining pattern for bcl-2 was similar to that of BPH. [1999]

Expression of mRNA for heregulin and its receptor, **ErbB-3** and ErbB-4, in human upper gastrointestinal mucosa. [1998]

CONCLUSION: These results indicate that the inhibition of HT-29 cell growth by t10c12 may be induced via its modulation of **ErbB3** signaling leading to inhibition of Akt activation. [2005]

In conclusion a) the results of the present study demonstrate the presence of c-erbB-2 and **c-erbB-3** oncoprotein expression (overexpression) in melanoma and breast carcinoma, and b) oncogene receptor directed immunotherapy, as part of a more individualized anti-cancer treatment, represents a potentially valuable targeted treatment for the future. [1997]

In conclusion, our data demonstrate new aspects of the phenotype and the biological behavior of CCSST and reveal **ERBB3** to be a useful diagnostic marker. [2004]

HER3 mRNA was detected in 55% of primary or metastatic human colorectal carcinomas but in only 22% of normal colon mucosa and 32% of normal liver samples. [1991]

In this work, we altered the levels of ErbB2 and **ErbB3** receptors, individually and in combination, by using 6-finger and 12-finger synthetic zinc finger protein artificial transcription factors (ATFs) in an epidermoid squamous cell carcinoma line, A431. [2005]

To verity the role of metastasis-related nm23 genes in carcinogenesis and progression of ovarian carcinoma, we analyzed the mRNA levels of the nm23 genes of both isoforms, -H1 and -H2, together with those of the epidermal growth factor receptor, the c-erbB-2, and the **c-erbB-3** genes in 45 ovarian carcinomas and 5 benign cystadenomas. [1994]

Wild type HRGbeta egf domain displayed on phage was properly folded as evidenced by its ability to bind **ErbB3** and ErbB4 receptor-IgG fusion proteins with affinities close to those measured for bacterially produced HRGbeta egf domain. [1998]

125I-rHRG beta 1(177-244) could be chemically cross-linked to a 170-180 kDa protein in erbB3-transfected fibroblasts, and the cross-linked product could be immunoprecipitated with antibodies specific for **ErbB3**. [1994]

The activation of mHOG by ErbB2 or **ErbB3** has not been reported, and may contribute to the unusual phenotype. [1997]

Basal proliferation in the absence of growth factors was also inhibited by **GW572016** to a greater extent than **ZD1839**, suggesting that low level HER2/**HER3** activation perhaps by an autocrine pathway contributes to the proliferation signal. [2005]

Role of the N-terminus of epidermal growth factor in ErbB-2/**ErbB-3** binding studied by phage display. [2002]

High **c-erbB-3** protein expression is associated with shorter survival in advanced non-small cell lung carcinomas. [1997]

In conclusion, high **c-erbB-3** expression in advanced non-small cell lung carcinomas might be an adverse prognostic factor. [1997]

This finding suggests that **c-erbB-3** might be a potential target for molecular therapy in advanced non-small cell lung carcinomas. [1997]

top

These findings suggest that HRG and its receptors, **ErbB-3** and ErbB-4 may be physiologically significant in the human upper GI mucosa, especially in duodenum, and that ErbB-4 may contribute to the growth of gastric cancer. [1998]

Trans-10,cis-12, not cis-9,trans-11, conjugated linoleic acid decreases **ErbB3** expression in HT-29 human colon cancer cells. [2005]

We analyzed the influence of **ErbB3** expression on pancreatic cancer cell response to erlotinib treatment. [2007]

The effects of RNA inhibition of **ErbB3** on sensitivity to erlotinib treatment were evaluated in AsPC-1 pancreatic cancer cells. [2007]

Cell migration on collagen was decreased when ErbB2 was down-regulated, yet migration on laminin was significantly increased with **ErbB3** overexpression. [2005]

We conclude that ErbB2 and **ErbB3** are expressed in T(84) cells and are functionally coupled to inhibition of calcium-dependent chloride secretion. [1999]

As there is no evidence of gene amplification, we decided to examine the control of **c-erbB-3** transcription in overexpressing cells. [1993]

Expression of the **c-erbB-3** protein in normal human adult and fetal tissues. [1992]

The level of expression of the mRNA for **c-erbB-3** was also examined in extracts of a selection of fetal tissues. [1992]

Transfection of MTLn3 cells and MDA-MB-435 cells with expression vectors for ErbB family members ErbB1, ErbB2 and **ErbB3** also significantly enhanced EF-induced migration. [2007]

Its receptor is **HER-3**, which is up-regulated on the membranes of keratinocytes lining the edge of the wound and on keratinocytes that had migrated towards the centre of the wound. [2002]

1,25-(OH)2D3 caused a marked increase in the cellular contents of ErbB1, ErbB2, and **ErbB3** proteins. [1999]

By immunoprecipitation followed by Western blotting we showed that ErbB1/**ErbB3** heterodimers are the major mitogenic signaling entity in 1,25-(OH)2D3-stimulated cells. [1999]

Frequency and significance of **c-erbB-3** overexpression in lung cancers have not been reported previously. [1997]

The identification of a NRG-1alpha/ErbB2/**ErbB3** autocrine loop raises the possibility that interruption of this loop may have therapeutic potential in lung cancer. [2004]

Epidermal growth factor receptor, c-erbB2 and **c-erbB3** receptor interaction, and related cell cycle kinetics of SK-BR-3 and BT474 breast carcinoma cells. [2001]

Listeria monocytogenes produces a pro-invasive factor that signals via ErbB2/**ErbB3** heterodimers. [2005]

CONCLUSIONS: The interaction between Listeria and collagen type I produces, next to the 13-mer peptide, at least another pro-invasive factor that signals via ErbB2/**ErbB3** heterodimers. [2005]

ErbB3 protein expression was low in all gliomas analyzed. [2004]

On multivariate analysis, positive margins, lymphatic invasion, and **HER-3** expression were significant predictors of survival outcome. [2008]

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In contrast, **c-erbB-3** mRNA levels were 3.5-fold lower ($p < 0.01$) in the esophageal cancers than in the normal tissues. [1999]

Concomitant analysis of the epidermal growth factor receptor family in esophageal cancer: overexpression of epidermal growth factor receptor mRNA but not of c-erbB-2 and **c-erbB-3**. [1999]

The analysis of human tumors revealed a significant relationship between alpha6beta4 and **ErbB-3** in P-Akt-positive and ERbeta1-negative breast cancers derived from patients with lower disease free survival. [2008]

In multivariate analysis, tumor grade and **HER3** expression were significantly predictive of overall survival. [2006]

Skin and A431 cells expressed more **ErbB3** than did keratinocytes. [2001]

Another gene, **ERBB3**, is involved in oligodendrocyte differentiation. [2005]

Schizophrenia is not associated with the functional candidate gene **ERBB3**: Results from a case-control study. [2007]

Two SNPs in **ERBB3** (rs773123 and rs2271188) were polymorphic in our samples, neither of which showed significant evidence of association with the illness (P = 0.639 and 0.561, respectively). [2007]

We report that ligand-induced degradation of internalized ErbB-1, but not **ErbB-3**, is mediated by transient mobilization of a minor fraction of c-Cbl into ErbB-1-containing endosomes. [1998]

Intriguingly, loss of ErbB2 signaling is accompanied by a decrease in the phosphotyrosine content of **ErbB3**. [2003]

We have generated peptide-specific antisera that recognizes the 160-kDa **HER3** protein when transiently expressed in COS cells. [1990]

In further support of this hypothesis, we provide evidence using reverse transcription-PCR analysis that expression of the ErbB-2 and ErbB-4 receptors, but not ErbB-1 or **ErbB-3**, is deregulated in medulloblastoma compared with normal developing cerebellum. [1998]

In addition, NDF caused a down-regulation of ErbB2 but not of **ErbB3**. [1997]

Binding interaction of the heregulinbeta egf domain with **ErbB3** and ErbB4 receptors assessed by alanine scanning mutagenesis. [1998]

Similar staining patterns were observed for c-erbB2 and **c-erbB3** in the respiratory nasal epithelium. [2000]

In normal endometrium, the **c-erbB-3** receptor was weakly expressed in both phases. [1999]

Roles of **ErbB-3** and ErbB-4 in the physiology and pathology of the mammary gland. [1997]

The use of the yeast two hybrid system to evaluate **ErbB-3** interactions with SH2 domain containing proteins. [1998]

c-erbB3 product is moderately expressed in gastric mucosa, especially in parietal cells. [1993]

The functions of ErbB2, **ErbB3**, and ErbB4 are also regulated by endocytosis to some extent, although the current knowledge of these processes is sparse. [2008]

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Expression of **c-erbB-3** did not significantly differ among placental and gestational trophoblastic disease tissues and trophoblastic cell types except for significantly increased expression in choriocarcinoma as compared with cytotrophoblasts of partial mole (P = 0.02). [2000]

ErbB3 was localized to the cytosol of rat conjunctival goblet cells. [2008]

ErbB3 was detected in only one of the meningiomas analyzed. [2004]

Membrane proteins are solubilized via Triton X-100 lysis and the receptor is captured in ELISA wells coated with ErbB2-specific antibodies with no cross-reaction to **ErbB3** or ErbB4. [1996]

Here, we demonstrate that ErbB2, **ErbB3** and ErbB4 are expressed in cultured human melanocytes. [2005]

HRG, **ErbB-3** and ErbB-4 mRNA were detected in esophagus, stomach and duodenum and the highest expression was found in duodenum. [1998]

HER3 expression was associated with decreased survival in proportional hazard modeling, including the International Federation of Gynecology and Obstetrics (FIGO) stage, histologic grade and type, residual disease, and age. [2006]

Immunoblot analysis showed that neither genistein nor daidzein decreased the protein levels of either of the epidermal growth factor receptors, ErbB2 or **ErbB3**. [2005]

ErbB3 is unique among other members of the receptor tyrosine kinase family of growth factor receptors in that its kinase domain is enzymatically impaired. [1999]

Heregulins (neuregulins) are a family of proteins known to interact and activate the receptor tyrosine kinases ErbB2 in association with **ErbB3** or ErbB4. [1996]

ErbB2 is a member of the ErbB/type I family of receptor tyrosine kinases which also includes epidermal growth factor receptor, **ErbB3** and ErbB4. [1996]

To test the feasibility of this strategy we targeted **ErbB3**, a member of the ErbB family of tyrosine kinase receptors, using this strategy. [2006]

A comparison of the deduced amino acid (aa) sequences of human and rat **ErbB3** was made, and the effects of certain aa substitutions in the putative protein tyrosine kinase domain were considered. [1995]

ErbB3 was constitutively tyrosine phosphorylated in both the parental AU565 cells and in the erbB2 nonexpressing cells. [1998]

Epidermal growth factor receptor tyrosine kinase mutations are the crux of targeted therapies, whereas epithelial-mesenchymal transitions and **HER3** mRNA levels are promising ancillary markers for treatment with epidermal growth factor receptor tyrosine kinase inhibitors. [2006]

The antibodies recognize **c-erbB-3** by immunoprecipitation and Western blotting of lysate prepared from a cell line engineered to overexpress the protein. [1993]

The monoclonal antibody was found to modestly but significantly stimulate the anchorage-independent cloning efficiency of the breast tumour cell lines BT483 and T47D, both of which express the **c-erbB3** protein. [1994]

Consequently, we became interested in using RNAi to determine the function of aberrantly expressed **ErbB3** in the KAS-6/1 human myeloma cell line. [2002]

A monoclonal antibody to the human **c-erbB3** protein stimulates the anchorage-independent growth of breast cancer cell lines. [1994]

Efficient NDF-induced phosphorylation of **ErbB-3** was strictly ErbB-2 dependent in the breast tumor cell lines T47D and MCF7, while it was largely ErbB-2 independent in MCF10A and OVCAR3 cells. [1995]

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CONCLUSION: We postulate that **HER-3** is critically involved in colorectal tumourigenesis and its expression/phosphorylation might be of prognostic significance. [2007]

Although **HER-3** signalling is known to be implicated in colorectal carcinogenesis, the significance of its expression, localisation and phosphorylation remains elusive. [2007]

The aim of this study was to analyze the expression and localization of **ErbB3** in prostate tissues and prostate cancer cell lines. [2006]

ErbB3 subcellular localization was studied by Western blot analysis in LNCaP, 22Rv1, PC-3, and DU145 prostate cancer cell lines. [2006]

In this report, we describe the states of association of ErbB2 and their relationship to local **ErbB3** density and lipid rafts based on quantitative fluorescence microscopy of SKBR-3 breast cancer cells. [2002]

A series of 346 patients with primary operable breast cancer and a series of 145 patients with advanced breast cancer were investigated for **c-erbB-3** protein expression using the monoclonal antibody RTJ1. [1996]

c-erbB-3, A recently identified member of the type I tyrosine kinase receptor family, has been shown to be overexpressed in invasive ductal carcinoma of breast. [1997]

All-trans (ATRA) and 9-cis retinoic acid (9cRA) reduce c-erbB-1 protein to 50-100%, c-erbB-2 to 20-30%, and **c-erbB-3** to 10-50% of control, depending on the concentration, respectively, without influencing the tyrosine phosphorylation status. [1998]

17beta-estradiol only slightly decreased basal tyrosine phosphorylation of ErbB-2 and **ErbB-3**. [1998]

c-erbB-3 protein expression in human breast cancer: comparison with other tumour variables and survival. [1994]

NDF-induced phosphorylation of ErbB-2 and **ErbB-3** was found in the breast epithelial cell line MCF10A, the breast tumor cell lines T47D and MCF7, and the ovarian tumor cell line OVCAR3. [1995]

There was no relationship between the presence or the amount of ErbB1, phospho-ErbB1, phospho-ErbB2, **ErbB3**, ErbB4, phospho-Akt, and Akt or the ability of lapatinib to inhibit phospho-ErbB1 in these cell lines compared to those that did not respond to lapatinib. [2005]

Transactivation of **ErbB-3** correlates with heterodimer formation and is reflected in receptor phosphorylation and the transregulation of ligand affinity. [1996]

Twenty-four per cent of carcinomas had membrane 21N staining, and 12% presented strong and generalised positivity ( ). **c-erbB-3** protein expression was significantly associated only with that of c-erbB-2 (P = 0.05), whereas 21N positivity was significantly associated with small tumour size (P = 0.02) and ductal histotype (P = 0.04). [1994]

Only expression of c-erbB-2 was found to correlate with the increase in the tumour stage, while co-expression of c-erbB-2, **c-erbB-3** and c-erbB-4 was found to correlate with the patient survival time in 25% of the carcinomas examined. [1997]

Membranes from SMDF-transfected cells stimulated tyrosine phosphorylation of the beta-heregulin receptor **ErbB3** in Schwann cells. [1998]

Interestingly, Western blot analysis of cytoplasmic and nuclear subcellular fractions showed that **ErbB3** nuclear localization was more prevalent in hormone-sensitive prostate cancer cell lines (LNCaP and 22Rv1) compared with hormone-insensitive cell lines (PC-3 and DU145). [2006]

ErbB2 and **ErbB3** receptor tyrosine kinases have been associated with the development of human colon cancer, and the expressions of both receptors are high in HT-29 cells. [2005]

Stimulation of Schwann cell **ErbB3** receptor phosphorylation by SMDF was not affected by inhibition of Schwann cell heparan sulfate proteoglycan synthesis. [1998]

Model results applied to experimental data on ErbB 1, ErbB2 and **ErbB3** phosphorylation in H292 human lung carcinoma cells support a hypothesis that key dephosphorylation activity for these receptors occurs largely in an intracellular, endosomal compartment rather than at the cell surface plasma membrane. [2006]

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Following LAQ824 treatment, the cell membrane association, autotyrosine phosphorylation, and colocalization of Her-2 with **HER-3** also declined. [2003]

Expression of **c-erbB-3** and c-erbB-4 proteins in papillary thyroid carcinomas. [1996]

Coexisting overexpression of epidermal growth factor receptor, c-erbB-2, **c-erbB-3**, and c-erbB-4 was demonstrated in 36 (64%) of 56 papillary thyroid carcinomas. [1996]

When present in the specimen, **c-erbB-3** and cerbB-4 immunopositive staining was seen in some of the oral surface epithelial cell layers (basal, intermediate and/or superficial) as well as the tumour islands. [1997]

CONCLUSIONS: **ErbB3** nuclear localization discriminates normal from malignant prostate tissues and between tumors from hormone-sensitive versus hormone-refractory prostate cancer. [2006]

The correlation between **ErbB-3** and distant metastasis was good. [2001]

A discrete three-amino acid segment (LVI) at the C-terminal end of kinase-impaired **ErbB3** is required for transactivation of ErbB2. [1999]

Heregulin binding to **ErbB3** induces formation of a heterodimeric complex with ErbB2, and this results in transactivation of the ErbB2 kinase. [1999]

We conclude that formation of a functional ErbB2-ErbB3 signaling complex requires the presence of a specific structural feature within the **ErbB3** cytoplasmic domain and suggest that ErbB2 transactivation results from a physical interaction between the cytoplasmic domains of these receptors. [1999]

RESULTS: Immunohistochemistry analysis of prostate cancer tissues revealed that >90% of prostate cancer tissues displayed cytoplasmic **ErbB3** staining. [2006]

Interactions and effects of **ErbB3** were studied in detail in adenocarcinoma lines H441 and H1373. [2003]

The pattern of **c-erbB-3** gene product was studied in 91 advanced gastric carcinomas, adjacent hyperplastic mucosa, intestinal metaplasia and dysplasia and in normal controls, using immunohistochemistry in archival material. [1993]

The expression of the c-erbB-2, **c-erbB-3** and c-erbB-4 members of the epidermal growth factor receptor family was examined in 16 fresh frozen tissue specimens of SCCHN using avidin-biotin complex immunohistochemistry, with monoclonal and/or polyclonal antibodies directed against each. [1997]

All the normal control oral mucosa showed the same pattern of staining for c-erbB-2, **c-erbB-3** and c-erbB-4 found in the epithelium located near the carcinomas. [1997]

Exposure to pAb alone reduced total ErbB2 protein, disrupting **ErbB3** transactivation, leading to a marked inhibition of p-Akt; however, survivin protein levels remained unchanged and apoptosis only increased slightly. [2005]

Formation of a high affinity heregulin binding site using the soluble extracellular domains of ErbB2 with **ErbB3** or ErbB4. [1998]

In contrast, heterodimeric **ErbB3**/4-IgG, as well as homodimeric ErbB3-IgG or ErbB4-IgG, contained only low affinity HRG binding sites. [1998]

c-erbB-3 protein expression in ductal carcinoma in situ of the breast. [1997]

Overexpression of **c-erbB-3** in various stages of human squamous cell carcinomas. [1998]

c-erbB-3 and c-erbB-4 protein expression was analyzed using immunohistochemistry in 138 fresh-frozen thyroid tissue samples from 106 patients, including 56 cases of papillary thyroid carcinoma. [1996]

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EXPERIMENTAL DESIGN: Immunohistochemistry of **ErbB3** was done on prostate cancer tissue sections from 143 patients and on a tissue microarray containing 390 cores of radical prostatectomy-derived specimens representing normal, prostatic intraepithelial neoplasia, and malignant tissues from 81 patients. [2006]

Cell cycle activation in lung adenocarcinoma cells by the **ErbB3**/phosphatidylinositol 3-kinase/Akt pathway. [2003]

Further complexity is added due to the existence of an oncogenic receptor that enhances and stabilizes dimerization but has no ligand (ErbB-2), and a receptor that can recruit novel SH-2-containing proteins, but is itself devoid of kinase activity (**ErbB-3**). [1997]

Specifically, ligand-dependent ErbB2 transactivation requires a discrete three-amino-acid segment, located at the C-terminus of ErbB family members **ErbB3**, ErbB4, and the epidermal growth factor receptor. [2001]

In this study, expression of the **c-erbB-3** protein was examined in 57 cases of pure ductal carcinoma in situ of the breast (DCIS) by immuno-cytochemical methods. [1997]

In conclusion, we have shown that quercetin inhibits cell growth and induces apoptosis in colon cancer cells, and that this may be mediated by its ability to down-regulate ErbB2/**ErbB3** signaling and the Akt pathway. [2005]

It is conceivable that combinatorial receptor interactions diversify signal transduction and confer double regulation, in cis and in trans, of the superior mitogenic activity of the kinase-defective **ErbB-3**. [1996]

The **c-erbB-3** protein was found in normal epithelial cells throughout the GI tract, in squamous epithelium of the oropharanyx and oesophagus, in the parietal cells of the stomach, and in the surface enterocytes of the small and large bowel. [1993]

Mapping a heparin binding site on **ErbB-3** epidermal growth factor receptor. [2001]

BACKGROUND: To elucidate the relationship between the expression of epidermal growth factor receptor family members (ErbB-1, neu/ErbB-2, **ErbB-3**, and ErbB-4) and tumor recurrence. [2001]

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